EFFECT OF VALSARTAN ON SUPPRESSION OF NEOINTIMAL HYPERPLASIA AFTER DRUG-ELUTING STENTS AND ON REGRESSION OF NATIVE PLAQUE VOLUME: THE VAL-SUPPRESS RANDOMIZED CONTROLLED TRIAL

i2 Poster Contributions
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Authors: Duk-Woo Park, Yangsoo Jang, Ki-Bae Seung, Hyeon-Cheol Gwon, Seung-Jea Tahk, Chang Hoon Lee, Gyung-Min Park, Yong-Giun Kim, Ki Won Hwang, Jung-Min Ahn, Hae Geun Song, Won-Jang Kim, Jong-Young Lee, Soo-Jin Kang, Seung-Whan Lee, Young-Hak Kim, Cheol Whan Lee, Seong-Wook Park, Seung-Jung Park, Asan Medical Center, Seoul, South Korea, Yonsei University Medical Center, Seoul, South Korea

Background: Accumulating evidence suggests that angiotensin-converting enzyme activity may in part mediate neointimal hyperplasia after stenting and progression of atherosclerotic disease. We hypothesize that use of valsartan might suppress intimal hyperplasia after coronary stenting and might influence regression of native atheromatous plaque.

Methods: The Val-SUPPRESS trial is a prospective, randomized, multi-center, open-labeled trial. We randomly assigned a total of 220 patients with de novo coronary disease receiving paclitaxel-eluting or zotarolimus-eluting stents to receive valsartan 160 mg daily with open treatment or not. The primary end point was an in-stent late loss of the stented segments at 8-month angiographic follow-up. Secondary end points included serial changes of percent atheroma volume of non-stented, intermediate lesions (i.e., most diseased 10-mm segment) by intravascular ultrasound, and clinical outcomes (death, myocardial infarction, and repeat revascularization).

Results: A total of 220 patients were randomized 1:1 to received valsartan treatment (n=108) or not (n=112). Eight-month angiographic follow-up was performed in 86% of overall patients. The primary end-point of in-stent late loss was similar in patients receiving valsartan and in those not receiving valsartan (0.52±0.40 and 0.54±0.50, P=0.64). The rate of angiographic restenosis and the combined and individual clinical endpoints were also similar between 2 groups. However, the mean (SD) change in atheroma area in the most diseased 10-mm subsegment significantly differ (-1.24±2.22 mm² in the valsartan group and 0.17±1.50 mm² in the no-valsartan group, P=0.01). Change in regression of atheroma volume was also significantly higher in the valsartan group than in the no-valsartan group (-7.86±11.00 mm³ and -2.10±9.61 mm³, P=0.05).

Conclusions: Valsartan treatment did not significantly reduce neointimal hyperplasia after drug-eluting stents implantation. However, valsartan treatment resulted in significant regression of atherosclerosis in native coronary disease. Further studies are needed to determine the effect of the observed changes on clinical outcome.